Longitudinal genetic study of verbal and nonverbal IQ from early childhood to young adulthood

LONGITUDINAL GENETIC STUDY OF VERBAL AND NONVERBAL IQ FROM EARLY CHILDHOOD TO YOUNG ADULTHOOD

Rosa A. Hoekstra*, Meike Bartels, Dorret I. Boomsma

Department of Biological Psychology, Vrije Universiteit, Van der Boechorststraat 1, 1081 BT Amsterdam, the Netherlands.

* To whom correspondence should be addressed.

Phone: +31 20 5988363; Fax: +31 20 5988832; E-mail: Ra.Hoekstra@psy.vu.nl.

Acknowledgements:

Financial support was given by The Netherlands Organization for Scientific Research (NWO 575-25-006) & (NWO/SPI 56-464-14192). Dr Bartels is financially supported by NWO (VENI 451-04-034). We are indebted to all the participating twin families. Furthermore we would like to thank Hanneke Hulst for her assistance in the data collection and data management.

Published as:

Hoekstra, R.A., Bartels, M., Boomsma, D.I., 2007. Longitudinal genetic study of verbal and nonverbal IQ from early childhood to young adulthood. Learning and Individual Differences, 17, pp 97-114.

LONGITUDINAL GENETIC STUDY OF VERBAL AND NONVERBAL IQ FROM EARLY CHILDHOOD TO YOUNG ADULTHOOD

Abstract

In a longitudinal genetic study we explored which factors underlie stability in verbal and nonverbal abilities, and the extent to which the association between these abilities becomes stronger as children grow older. Measures of verbal and nonverbal IQ were collected in Dutch twin pairs at ages 5, 7, 10, 12 and 18 years. The stability of both verbal and nonverbal abilities was high, with correlations over time varying from .47 for the 13-year time interval up to .80 for shorter time intervals. Structural equation modeling showed increasing heritability with age, from 48% (verbal) and 64% (nonverbal) at age 5 to 84% and 74% at age 18. Genetic influences seemed to be the driving force behind stability. Stability in nonverbal ability was entirely explained by genes. Continuity in verbal abilities was explained by genetic and shared environmental effects. The overlap between verbal and nonverbal abilities was fully accounted for by genes influencing both abilities. The genetic correlation between verbal and nonverbal IQ increased from .62 in early childhood to .73 in young adulthood.

Introduction

General cognitive ability, or intelligence, is one of the best studied areas in behavior genetics (see for reviews Bouchard, Jr. & McGue, 2003; Deary, Spinath, & Bates, 2006; Plomin & Spinath, 2004). Twin family and adoption studies have examined genetic and environmental influences on cognition at several time points across the life span. It is well established that genetic factors increase in importance over the life time, whilst shared environmental influences diminish. The heritability of general cognitive ability in infancy is estimated at about 20% (Fulker, DeFries, & Plomin, 1988; Wilson, 1983; Bishop et al., 2003; Petrill et al., 2004; Spinath, Ronald, Harlaar, Price, & Plomin, 2003), increases to about 40% in middle childhood (e.g. Bartels, Rietveld, Van Baal, & Boomsma, 2002) and may be as high as 80% in adulthood (e.g. Posthuma, De Geus, & Boomsma, 2001; Rijsdijk, Vernon, & Boomsma, 2002). In parallel, shared environmental influences explain about half of the variance in intelligence in young children (Bartels et al., 2002; Spinath et al., 2003), decrease in importance at later ages in childhood (Bartels et al., 2002), and become non significant by adolescence (e.g. Posthuma et al., 2001; Rijsdijk et al., 2002; Scarr & Weinberg, 1983).

Stability of general cognitive ability

Longitudinal studies show that general cognitive ability is a highly stable trait. A 68-year follow-up of almost 500 people showed a stability coefficient of .66 between IQ scores on a test taken at age 11 years and 79 years (Deary, Whiteman, Starr, Whalley, & Fox, 2004). Cognitive ability in childhood (age 5 – 12) shows similar stability (Bartels et al., 2002), but tests conducted at very young ages may be less predictive of cognitive

abilities in later life (Bishop et al., 2003; Petrill et al., 2004), although these results have been challenged. The lack of prediction may stem from the fact that traditional measures of infant IQ, such as the Bayley Mental Development Index, are poor predictors of later IQ scores (Boomsma, 1993). Measures of infant cognitive function, such as habituation and novelty preference seem more predictive of later IQ (Bornstein & Sigman, 1986). DiLalla et al. (1990) measured novelty preference in twins 7, 8 and 9 months. Mid-twin scores were regressed on mid-parent WAIS-IQ and showed significant heritability at 9 months. Spinath et al. (2003) assessed verbal and nonverbal abilities in a longitudinal twin study at age 2, 3, and 4 years and reported 2-year stability coefficients ranging from .36 to .49. The authors of this study also performed a principal component analysis to derive a general intelligence (or "g") factor. The 2-year stability of g was found to be .60, suggesting that general cognitive ability can also be measured reliably in early life.

Developmental mechanisms underlying stability of general cognitive ability

Longitudinal twin and family studies enable disentangling genetic and environmental influences on the stability of cognitive abilities over time. The genetic and environmental influences may exert their effect on stability following different developmental mechanisms. Firstly, the same genetic or environmental influences may affect IQ throughout development, although their relative importance can change over time. This structure suggests an underlying factor (genetic or environmental) that influences cognitive ability at each time point and accounts for stability of intelligence over time. This type of developmental structure in genetic modeling is modeled as a common factor (Martin & Eaves, 1977; see Bartels et al., 2002 for a recent application).

Secondly, genetic or environmental influences may exert their effects by carrying over part of prior experiences to subsequent ages, together with new influences, or innovations, at each occasion. In this pattern, the influences on intelligence at successive ages are causally linked, so that each new event builds upon earlier experiences. Stability of intelligence over time is explained by the part of earlier influences that is transmitted to subsequent ages. Innovations, e.g. new genes that are expressed, can enter at each age. This developmental pattern is referred to as a transmission structure, or simplex model (Boomsma & Molenaar, 1987; see Bartels et al., 2002 for a recent application). This model is suggested when the phenotypic correlations decrease with longer time intervals (Jöreskog, 1970). Lastly, genetic and environmental influences may be specific to a certain time point only and not exert effects on the continuity of cognitive ability. These effects are referred to as age specific affects.

Different research groups have conducted twin and adoption studies of cognitive development. We focus here on the most recent findings of twin and adoption studies spanning a long time of development. The Louisville Twin Study (LTS), initiated nearly 50 years ago, includes almost 500 twin pairs and their siblings who have participated in a longitudinal study of cognitive development from age 3 months through 15 years (Wilson, 1983). Data from this study suggest that the continuity of cognitive ability is largely explained by genetic and shared environmental effects (Eaves, Long, & Heath, 1986), whilst non-shared environmental effects are occasion specific. The Colorado Adoption Project (CAP) has collected data on adopted children and their adoptive and biological parents and on non-adoptive (control) families. Reports up to now include data on cognitive development spanning age 1 to 16 years (Petrill et al., 2004). In this

longitudinal data set, stability in general cognitive ability was mainly accounted for by genetic effects. The genetic stability was accounted for by a common facture structure. Shared environmental effects were not significant, whereas non-shared environmental influences were mainly age specific. Bishop et al. (2003) studied cognitive development from age 1 to 12 years in a combined sample of the above mentioned CAP study and a longitudinal twin sample. They reported a transmission structure for genetic influences in early ages of development, changing into a common factor structure in later childhood. These genetic effects accounted for most of the stability in cognitive ability. Furthermore, a small shared environmental effect was found, that contributed to stability mainly from infancy through early childhood via a common factor pattern. Non-shared environmental influences were mainly age specific but also accounted for some stability in middle childhood. Bartels et al. (2002) studied cognitive development from age 5 to 12 years in a longitudinal twin sample from the Netherlands, overlapping with the sample used in the current paper. They reported a common factor structure for genetic influences, accounting for stability in total IQ at all ages. Shared environmental effects influenced stability as well as change via a common factor structure and age specific influences, whilst non-shared environmental influences were only age specific. The overall picture that can be drawn from these studies is that stability in cognitive ability is mainly accounted for by genetic effects. The non-shared environment is only of importance for effects specific to each time point and does not contribute to stability of cognitive abilities.

Specific cognitive abilities

Although genetic and environmental effects on the development of general intelligence are well documented, less is known about the development of specific cognitive abilities. A hierarchical organization of cognitive abilities is now widely recognized. A general cognitive factor accounts for about 50% of the variance in a broad variety of cognitive tests (Deary, 2001; Carroll, 1993). When this variance is taken into account, the remaining variance tends to cluster together into separable group factors of intelligence. Often, cognitive abilities are separated into verbal and nonverbal abilities (e.g. Wechsler intelligence scales verbal IQ (VIQ) and performance IQ (PIQ), Wechsler, 1997), or into more specific factors encompassing verbal comprehension, perceptual organization, working memory, and processing speed (Wechsler, 1997).

In adults, verbal abilities appear to be somewhat more heritable than nonverbal abilities. In two twin studies in young adults (Rijsdijk et al., 2002) and a sample of young and middle aged adults (Posthuma et al., 2001) heritability estimates for VIQ and PIQ were 84% and 85% for VIQ, and 68% - 69% for PIQ. In the latter twin sample (Posthuma et al., 2003), verbal comprehension was found to be somewhat more heritable (84%) than perceptual organization (68%), working memory (65%) and processing speed (63%). The Hawaii Family Study of Cognition, including data from 1816 families from American/European or Japanese ancestry, is one of the largest samples in which familial transmission of special cognitive abilities has been studied (DeFries et al., 1979). The midparent-offspring resemblance in both samples was higher for verbal (.48 - .54) and spatial (.60 - .42) abilities than for perceptual speed (.41 - .34) and memory (.31 - .18) factors (cited from Alarcón, Plomin, Corley, & DeFries, 2003). Two twins-reared-apart studies (McGue & Bouchard, Jr., 1989; Pedersen, Plomin, Nesselroade, & McClearn,

1992) reported heritabilities of 57 - 58%% (verbal abilities), 71 - .46% (spatial abilities), 53 -.58% (perceptual speed) and 42 - .38% (memory). Similar to general cognitive abilities, the heritability of specific cognitive abilities seems to increase with age. Whilst the heritability of verbal and nonverbal abilities is about 25% in infants (Price et al., 2000) the heritability increases to about 40% in middle childhood (Rietveld, Dolan, Van Baal, & Boomsma, 2003). Results from the LTS sample (Wilson, 1986) showed increasing monozygotic twin correlations for VIQ scores from age 5 to 15 years, whilst the dizygotic twin correlations remained stable. This pattern suggests increasing heritability over time. The heritability also increased with age for PIQ, but the twin correlations were somewhat lower than for VIQ, suggesting a larger influence of the non-shared environment. The CAP project reported a heritability of verbal, spatial, memory and perceptual speed abilities varying from 6 to 31% in 4-year-olds (Rice, Carey, Fulker, & DeFries, 1989). These estimates increased to 19 – 35% in 7-year-olds (Alarcón et al., 2003), to 26 – 53% in 12-year-olds (Alarcón, Plomin, Fulker, Corley, & DeFries, 1998; Alarcón et al., 2003), and to 32-64% when the offspring was 16 years old (Alarcón, Plomin, Fulker, Corley, & DeFries, 1999). Longitudinal model fitting of the CAP data in 3 to 9 year old children (Cardon, 1994) suggested that genetic effects are of main importance for the stability in specific cognitive abilities, exerting their effects via a transmission structure.

With multivariate genetic analyses, the extent to which genetic or environmental influences account for overlap between specific cognitive abilities can be examined. Such studies have found that genetic correlations (r_g , the extent to which genetic effects on one trait correlate with genetic effects on another trait) among specific cognitive abilities are

substantial in adulthood (r_g ranging from .35 to .87, depending on the tests used, Posthuma et al., 2001; Rijsdijk et al., 2002; Posthuma et al., 2003) and in middle to late childhood (r_g varying from .27 to .79, Casto, DeFries, & Fulker, 1995; Alarcón et al., 1998; 1999). In contrast, a study in infancy found a genetic correlation between verbal and nonverbal abilities of around .30 (Price et al., 2000). These findings suggest that genetic effects on specific cognitive abilities are largely independent in infancy, and become increasingly more correlated in later stages of cognitive development (Price et al., 2000; Petrill, 1997; Petrill, Saudino, Wilkerson, & Plomin, 2001; Plomin & Spinath, 2002). However, these results are based on cross-sectional comparisons.

Aims of the present study

The current paper reports on a longitudinal twin study of cognitive development spanning early childhood to young adulthood. Factor analyses of the data assessed at the first measurement occasion (when the twins were 5 years old), revealed a verbal and a nonverbal factor (Rietveld, Van Baal, Dolan, & Boomsma, 2000). A longitudinal analysis of the first three assessments when the twins were respectively 5, 7, and 10 years old (Rietveld et al., 2003) showed that stability in verbal and nonverbal ability was mainly due to genetic effects. The non-shared environment contributed to age specific variance only. The genetic correlation between verbal and nonverbal factors increased slightly over the years, but was still low at age 10 (r = .25 at age 5, to r = .30 at age 10), and of similar magnitude as the genetic correlation reported by Price et al. (2000). The current report is a follow-up of this study and includes assessment of verbal and nonverbal abilities at age 12 and 18 years in the same sample. This study aims to 1) examine genetic

and environmental influences on verbal and nonverbal abilities at 5 time points spanning development from age 5 to 18 years; 2) Explore the developmental structure underlying stability in verbal and nonverbal abilities; 3) Examine to which extent genetic effects influence the overlap between verbal and nonverbal abilities and to test if there is an increase in this correlation over development, as suggested by previous cross-sectional studies.

Methods

Participants

This project is part of an ongoing longitudinal study into the development of intelligence and problem behavior. The study was initiated in 1992 with the recruitment of 209 5-year-old twin pairs from the Netherlands Twin register (NTR), kept by the Department of Biological Psychology at the VU University in Amsterdam (Boomsma, Orlebeke, & Van Baal, 1992; Boomsma et al., 2002). The twin families were selected on the basis of age, zygosity of the twins, and their place of residence. Mean age at the first measurement occasion was 5.3 years (sd = 0.2). At the second measurement occasion (mean age 6.8 years, sd = 0.2) 192 pairs of the initial sample completed the test protocol. Around the tenth birthday of the twins (mean age 10.0 years, sd = 0.1) 197 twin pairs participated in the third data collection. The fourth assessment (mean age 12.0 years, sd = 0.1) was completed by 192 twin pairs. Six years later, 122 twin pairs of the initial sample participated in the fifth measurement occasion (mean age 18.1 years, sd = 0.2). To increase the sample size on the fifth assessment, 64 additional twin pairs (mean age 18.3 years, sd = 0.1) were recruited via the NTR. Complete data on all 5 measurement

occasions were available for 115 twin pairs. No significant differences in verbal and nonverbal IQ at age 5 were found for subjects who did not wish to participate in one of the assessments at age 7, 10 or 12 years ($F(3, 203) = .663$, $p = .576$ for verbal IQ; $F(3, 205) = 1.660$, $p = .177$ for nonverbal IQ). However, subjects who continued to participate at age 18 had higher mean verbal ($F(1, 205) = 7.834$, $p= .006$, $d = .40$) and nonverbal ($F(1, 207) = 4.471$, $p =.036$, $d = .30$) IQ scores at age 5 as compared to subjects who did no longer take part when they were 18 years old. The vast majority of the twins still lived with one or both of their parents at age 18 years.

Of all twin pairs from the longitudinal sample, 42 were monozygotic males (MZM), 44 were dizygotic males (DZM), 47 monozygotic females (MZF), 37 dizygotic females (DZF), and 39 dizygotic twin pairs of opposite sex (DOS). For the same-sex twin pairs, zygosity was based on blood group polymorphisms (63 pairs) or DNA analyses (100 pairs). For the remaining twins, zygosity was determined by physical resemblance assessed by an experienced test administrator (4 pairs) or by discriminant analyses of longitudinally collected questionnaire items (3 pairs). Of all newly recruited families that only participated at age 18, there were 13 MZM twin pairs, 12 DZM pairs, 16 MZF pairs, 9 DZF pairs and 14 DOS twin pairs. Zygosity determination in the same sex twins of this group was based on DNA analysis (37 pairs), blood group polymorphisms (7 pairs) or questionnaire items (7 pairs).

Procedures and intelligence tests

At ages 5 and 7, the twins participated in a study on the development of cognitive abilities and brain activity (Van Baal, Boomsma, & De Geus, 2001; Boomsma & Van

Baal, 1998). At both measurement occasions, the twins visited the university laboratory. While one of the twins participated in the electrophysiological experiment, the co-twin completed the intelligence test. At ages 10 and 12, the intelligence tests were conducted either at the twins' home or at the university, depending on the preference of the twin family. Most of the families preferred testing at home (around 70% at both ages). There were no significant differences in intelligence between the twins tested at home and the twins tested at the university (Bartels et al., 2002). At age 18 the children visited the university to complete the intelligence test as part of an extensive test protocol, including assessment of physical development and neuropsychological tasks. At all ages, the intelligence test was administered by experienced test administrators. At ages 5, 7, and 10, the test took approximately 1 hour to complete, and, at ages 12 and 18 the test took 1.5 hours to complete. At the end of each test protocol the twins received a present.

At age 5, 7, and 10 years, the children completed the Revised Amsterdamse Kinder Intelligentie Test (RAKIT, Bleichrodt, Drenth, Zaal, & Resing, 1984). The RAKIT is a Dutch psychometric intelligence test for children, with subtests covering a broad spectrum of intellectual abilities. The test is designed for children in the age of 4 to 11 years. The short version of the RAKIT was used, which has six subtests with age-appropriate items, measuring verbal and nonverbal abilities. Both the verbal and nonverbal IQ scores were based on the sum of three subtests scores, which were transformed into standardized scores. The standardization was based on a population sample of Dutch 6- to 11-year-old children; the norms for standardization were the same for boys and girls. For further details on this intelligence test, see Rietveld et al. (2003).

At age 12 the Dutch version of the Wechsler Intelligence Scale for Children–Revised (WISC-R, Van Haassen et al., 2006) was used. The complete test was conducted, encompassing 6 verbal and 6 nonverbal subtests. The WISC-R is an internationally used psychometric intelligence test and can be used from age 6 to age 16 years. Standardized verbal and nonverbal IQ scores were based on results of same-aged children in the Netherlands. The transformation from raw scores into standardized scores was based on the same norms for boys and girls.

At age 18 the Dutch version of the Wechsler Adult Intelligence Scale-third edition (WAIS-III, Wechsler, 1997) was administered. The twins completed 11 subtests, including 6 verbal and 5 nonverbal tests. The subtests were standardized for the appropriate age group, based on a population sample of same-aged subjects in the Netherlands. Standardization norms were the same across the sexes. Verbal and nonverbal ability scores were calculated as the mean subtest score on the 6 verbal, respectively the 5 nonverbal subtests. The concurrent validity of the RAKIT and the WISC-R is .86 (Pijl et al., 1984). The correlations between VIQ and PIQ scores measured with the WISC-R and the WAIS-R are high (.89 for VIQ, .76 for PIQ, Wechsler, 1981).

Statistical analyses

All analyses were carried out with structural equation modeling as implemented in the software package Mx (Neale, Boker, Xie, & Maes, 2006). To assess stability of verbal and nonverbal IQ over time, and the association between verbal and nonverbal abilities at all ages, phenotypic correlations were estimated in a saturated model. All data, regardless of the pattern of missingness, were analyzed using the raw data option in Mx.

By analyzing all data, any bias that may have been introduced by non-random drop out is corrected for (Little & Rubin, 2002). Twin correlations at each age and cross-twin/cross-age correlations were also estimated in the saturated model. These correlations give a first impression of the contribution of genetic and environmental effects on the variance of verbal and nonverbal abilities at each age, and on the etiology of stability of these traits over time. The cross-twin/cross-trait correlation (i.e. the correlation between verbal IQ in the one twin with nonverbal IQ in the co-twin) was also estimated at each age. These correlations give a first indication of the relative importance of genes and environment on the overlap between verbal and nonverbal abilities. Furthermore, it was tested whether the correlation patterns for verbal and nonverbal abilities were different across the sexes.

Genetic modeling

Monozygotic (MZ) twins are genetically identical at the DNA sequence level (but may show differences in gene expression due to e.g. differences in DNA methylation patterns (Jirtle and Skinner, 2007)). Dizygotic (DZ) twins share on average 50% of their segregating genes. This experiment of nature allows statistical modeling of twin data with the goal to attribute the observed variance into genetic and environmental contributions. Additive genetic variance (A) is the variance that results from the additive effects of alleles at each contributing genetic locus. Dominant genetic variance (D) is the variance that results from within locus interaction of the alleles at all contributing loci. Shared environmental variance (C) is the variance resulting from environmental effects common to both members of a twin pair. Non-shared environmental variance (E) is the variance caused by environmental influences that are not shared by members of a twin pair.

Estimates of the unique environmental influences also include measurement error. To take this source of variance into account, E is always specified in the model. Using twin data, the influence of C and D cannot be estimated simultaneously. However, comparing the twin correlations of MZ and DZ twins can give a first indication of what influences are important. If MZ and DZ twin correlations are similar, shared environmental influences are likely to be important. Conversely, a DZ twin correlation that is less than half the MZ twin correlation indicates dominance effects. Likewise, if MZ and DZ cross-twin/cross-age correlations are similar, shared environmental influences are expected to play a role in the stability over time. If MZ cross correlations are more than twice as large as DZ correlations, dominance effects may play a role in the continuity of cognitive ability. In this study, a model including influences of A, C, and E was tested, based on the twin correlations and cross correlations (see results-section).

Genetic modeling was performed using Mx (Neale et al., 2006), following several steps. The developmental pattern of verbal and nonverbal abilities from age 5 to 18 years was first examined in a Cholesky decomposition model. This approach decomposes the phenotypic relations into genetic, shared environmental and non-shared environmental contributions to the variance / covariance structure. All possible contributions are parameterized in the Cholesky decomposition; therefore it yields the best possible fit to the data. The model is descriptive rather than driven by any specific developmental hypothesis. However, it is useful to gain a first insight in what factors are important for the stability of verbal and nonverbal abilities. Furthermore it serves as a reference model to evaluate the fit of more parsimonious submodels. Based on the parameter estimates from the Cholesky decomposition, and on the findings from previous studies, several

submodels were tested including two developmental mechanisms: the common factor model and the transmission model. In the common factor model (Figure 1a), one underlying verbal factor and one underlying nonverbal factor are specified. These factors imply a continuous influence over time from the time of onset. The common verbal and nonverbal factors are allowed to correlate with each other. The transmission model (Figure 1b) represents a first-order autoregressive process. The covariances among the five measurement occasions are specified by the transmission of these effects to subsequent ages. Apart from the influences from prior time points, an innovation term unique to each measurement occasion can affect the variance. The total variance at each time point is the sum of the innovation effect and the age-to-age carry-over effect. Transmission and innovation factors are specified separately for verbal and nonverbal abilities. The genetic effects at age 5 and the innovation effects at subsequent time points on both abilities are allowed to correlate with each other at each time point.

Insert Figure 1a and 1b about here

The fit of the different developmental models and more parsimonious submodels was evaluated against the Cholesky model using χ^2 tests. The likelihood ratio test, which is the difference between minus twice the log likelihoods (-2 LL) of the two nested models under investigation, is distributed as a χ^2. The degrees of freedom (df) are given by the difference in the number of parameters estimated in the two models. A high increase in χ^2 against a low gain of degrees of freedom denotes a worse fit of the submodel compared to the full model. The most parsimonious model, with still a limited

χ^2, is chosen as the best fitting model. As the transmission model and the common factor model are not nested, it is impossible to use the χ^2 test to evaluate which model fits better. To select the best model, Akaike's information criterion (AIC = χ^2 – 2df) was computed. The model with the lowest AIC reflects the best balance between goodness of fit and parsimony. The best fitting parsimonious model was used to derive estimates of genetic, shared environmental and non-shared environmental effects on the variances and covariances of verbal and nonverbal abilities.

Results

The descriptives of verbal and nonverbal abilities at all five time points are given in Table 1. All variables were approximately normal distributed at all ages. Mean differences due to birth order or zygosity of the twins were absent. Mean verbal IQ scores were higher in boys than in girls ($\chi^2(5) = 14.919$, $p = .011$). The direction of the sex difference in mean nonverbal IQ ($\chi^2(5) = 13.836$, $p = .016$) varied per age group. In the genetic model fitting the means were specified separately for boys and girls, to account for the sex differences in the mean.

Insert Table 1 about here

Table 2 summarizes the phenotypic correlations for verbal (above diagonal) and nonverbal (below diagonal) abilities across time. Both verbal and nonverbal abilities show substantial stability over time; the phenotypic correlation over a 13-year time interval is .51 for verbal abilities and .47 for nonverbal abilities. However, the phenotypic

correlations decrease as the time intervals get larger. The last column in Table 2 gives the correlations between verbal and nonverbal abilities. As can be seen, these correlations increase with age. In early childhood verbal and non-verbal cognitive abilities are still largely independent (r = .33 - .35 at age 5, 7, and 10 years), and become increasingly more correlated in later stages of development (r = .58 at age 12, and .57 at age 18).

Insert Table 2 and 3 about here

Table 3 shows the twin correlations for the five zygosity groups estimated separately at each age. At all ages, and for both verbal and nonverbal abilities, the MZ correlations are higher than the DZ correlations, indicating genetic influences. The only exception to this pattern is verbal IQ at age 7, when MZ correlations are of the same magnitude as the DZ correlations. Apart from age 18, MZ correlations are not twice as high as DZ correlations, suggesting that shared environmental influences also play a role in familial resemblances. For both verbal and nonverbal abilities, the difference between MZ and DZ correlations tends to increase with age, suggesting that genetic influences become increasingly important with age. These patterns of correlations also suggest decreasing effects of the shared environment over time. Twin correlations in twins of opposite sex are similar to dizygotic same sex twins, yielding no indication that sex specific genes are of importance. The significance of sex differences in twin correlations was tested for both verbal and nonverbal IQ. Constraining MZ and DZ correlations to be the same across the sexes did not significantly worsen the model fit, neither for verbal abilities ($\chi^2(120) = 130.637$, p = .239) nor for nonverbal abilities ($\chi^2(120) = 139.144$, p =

.112). Therefore in subsequent modeling, data from male, female and opposite sex twins were pooled into 2 groups (MZ and DZ twins).

Insert Table 4 about here

Table 4 gives the MZ and DZ cross-twin/cross-age correlations for verbal and nonverbal abilities. MZ correlations are higher than DZ correlations, especially for nonverbal abilities, indicating genetic influence on stability. For verbal abilities, the MZ cross correlations are not twice as high as the DZ cross correlations, suggesting that for the stability of these cognitive abilities, shared environmental influences may also be of importance. Table 4 also shows the cross-twin/cross-trait correlation between verbal and nonverbal abilities at each time point. Apart from the first measurement occasion, MZ cross correlations are higher than DZ cross correlations, suggesting genetic effects on the overlap between verbal and nonverbal IQ.

Insert Table 5 and 6 about here

A series of developmental models was fitted to the data on verbal and nonverbal IQ (verbal abilities at age 5, 7, 10, 12, and 18 years of age, and nonverbal abilities at the same 5 time points). Table 5 gives the model fitting statistics for the Cholesky decomposition and the more parsimonious submodels. The parameter estimates from the Cholesky decomposition were inspected to get a first impression of the importance of the influence of A, C, and E to the variances and covariances between measures and between

twins. The Cholesky decomposition was used as the reference model to evaluate the fit of developmental models, incorporating different mechanisms for A, C, and E.

The parameter estimates based on the Cholesky decomposition are given in Table 6. These estimates, together with results from previous studies (Bartels et al., 2002; Petrill et al., 2004; Rietveld et al., 2003) both suggested that non-shared environmental influences are only of importance for explaining age specific variance in cognitive abilities, and do not have a significant role in explaining stability. Therefore, a model with solely age specific effects of the non-shared environment was applied. The parameters describing the shared environmental influences in the Cholesky decomposition showed the highest loadings on the first factor, and relatively low loadings on the other factors. Furthermore, the loadings were higher for verbal than for non-verbal abilities. This pattern suggests a common factor structure, with highest loadings on verbal IQ. Prior studies (Bartels et al., 2002; Rietveld et al., 2003; Bishop et al., 2003) also indicated that, if of importance, shared environmental influences would exert their effects via a common factor structure. Since one previous study (Bartels et al., 2002) in the same sample as the current study also found significant age specific effects of the shared environment, these effects were specified as well. The previous literature on the developmental mechanism underlying genetic influences is less clear-cut. Some studies report a transmission pattern (Cardon, Fulker, DeFries, & Plomin, 1992; Rietveld et al., 2003; Cardon, 1994), others found a common factor model (Bartels et al., 2002; Petrill et al., 2004), or a combination of these models (Bishop et al., 2003). In our data the Cholesky decomposition did not give a clear indication for a transmission structure (i.e. decreasing factor loadings with increasing time intervals) or a common factor

developmental pattern (i.e. high loadings on one factor). Therefore, both a transmission model and a common factor model including age specific effects were fitted to the data.

To summarize, two models were evaluated. The first model included a common factor structure together with age specific influences for A and C, combined with only age specific influences for E (model 2 in Table 5). In the second model, genetic effects were modeled in a transmission structure, shared environmental effects in a common factor structure including age specific effects, while non-shared environmental influences were again specified to only have age specific effects (model 3 in Table 5). Application of these submodels did not result in a significant deterioration of the fit compared to the Cholesky decomposition (model 2: $\chi^2(113) = 133.173$, $p = .095$; model 3: $\chi^2(111) = 122.085$, $p = .222$). To evaluate whether model 2 or model 3 showed a better fit to the data, AIC's were compared. Since the AIC was lowest for the model including a transmission structure for additive genetic influences (model 3), this model was chosen as the best model.

We next tested the significance of the loadings on the shared environmental factor separately for verbal abilities and nonverbal abilities. Constraining the factor loadings on the nonverbal common factor to be zero (model 4) did not lead to a significant drop in model fit ($\chi^2(6) = 9.773$, $p = .135$). Age specific shared environmental influences on nonverbal abilities were not significant either (model 5, $\chi^2(5) = 3.283$, $p = .656$). These results indicate that all shared environmental influences on nonverbal abilities could be omitted. Subsequently, the significance of the shared environmental common factor on verbal abilities was tested. Since previous studies reported diminishing influences of C with age, the significance of the loadings on the common factor at later time points was

tested first. The role of common factorial C on verbal abilities appeared to be non-significant at age 18 (model 6, $\chi^2(1) = 1.486$, p = .223), and at age 12 (model 7, $\chi^2(1) = 1.945$, p = .163). The influence of the common factor on age 10 however was of significant importance. Constraining this factor loading to be zero resulted in a significant deterioration of the model fit (model 8, $\chi^2(1) = 18.665$, p = <.001). Additionally, the significance of the shared environmental influences specific to each time point were tested. The age specific influences of C were not of significant importance at age 18 (model 9, $\chi^2(1) = 0.00$, p = 1.00), but were significant at age 12 (model 10, $\chi^2(1) = 4.761$, p = .029). Taken together, the most parsimonious model with still acceptable fit (model 9 in Table 5; illustrated in Figure 2) was a model with i) a transmission structure for additive genetic influences; ii) a shared environmental common factor structure at age 5, 7, and 10 and time specific shared environmental influences at age 5, 7, 10 and 12 years for verbal abilities only; iii) non-shared environmental influences that only exert age specific effects. The covariance between verbal and nonverbal abilities is entirely accounted for by genetic effects, which were allowed to correlate at each time point.

Insert Figure 2 and Table 7 and 8 about here

Based on the best fitting model, the contributions of A, C, and E on the variance and covariance of verbal and nonverbal abilities were calculated. The contribution of genetic influences is given by the matrix formula: $A = (I-B)^{-1}*X*R*X'*((I-B)^{-1})'$ where matrix B (dimension 10x10, for the 10 variables in the study) contains the genetic transmission parameters on its subdiagonal. The genetic innovation parameters are

modeled in matrix X (a diagonal 10x10 matrix). Matrix R is a 10x10 correlation matrix, in which the 5 within-age correlations between the genetic innovations of verbal and nonverbal abilities are estimated. Matrix I (10x10) is an identity matrix. Likewise, the contribution of the shared environmental influences is obtained by the matrix formula:

$C = Y*Y' + W*W'$

where matrix Y (10x10) contains the loadings on the common factor (constrained to be zero for the nonverbal abilities, and for verbal abilities at age 12 and 18 years), and matrix W (10x10) contains the age specific C influences on the diagonal (constrained to be zero for the nonverbal abilities, and for verbal abilities at age 18). The contribution of the non-shared environmental influences is given by the matrix formula: $E = Z*Z'$ where Z is a 10x10 diagonal matrix including the age specific influences of the non-shared environment.

The relative contribution of A, C, and E to the variance of verbal and nonverbal IQ are presented in Table 7. As indicated by the MZ and DZ twin correlations, additive genetic effects become increasingly important with age, especially for verbal abilities. The heritability of verbal abilities increases from 46% at age 5 to 84% at age 18. Shared environmental influences also play a role in variance in childhood verbal IQ, but become insignificant in adolescence. Non-shared environmental influences seem to become slightly less important over time, but the confidence intervals for these effects at the different ages overlap. Shared environmental effects do not play a role in explaining variance in nonverbal abilities. The additive genetic effects become somewhat more important in explaining variance in nonverbal IQ at later stages of development, the heritability rises from 64% at age 5 to 74% at age 18. Table 8 shows the relative

contribution of A, C, and E on the between-age covariance of verbal and nonverbal abilities. In early and middle childhood, the stability of verbal abilities is explained by both genetic and shared environmental influences. Between ages 5 and 10, shared environmental influences account for 22 – 37% of the covariance in verbal abilities. The remaining proportion of the covariance is explained by genetic effects that are transmitted to subsequent time points. At later stages of development (>10 years), shared environmental influences are no longer important, and the stability of verbal abilities is entirely accounted for by genetic effects. The non-shared environmental effects on verbal abilities only exert age specific influences and do not contribute to the stability of verbal IQ. The stability of nonverbal abilities is entirely explained by genetic effects. The non-shared environmental effects are only age specific. Lastly, the genetic correlations between verbal and nonverbal abilities are given in Table 9. The overlap between verbal and nonverbal abilities is entirely explained by genetic effects. Similar to the phenotypic correlations between verbal and nonverbal abilities (see Table 2), the genetic correlation increases slightly with age, from .62 at age 5 to .73 at age 18.

Insert Table 9 about here

Discussion

This study examined the genetic and environmental influences on verbal and nonverbal abilities between ages 5 and 18 years, investigated the developmental pattern underlying stability, and assessed the genetic correlation between verbal and nonverbal abilities at different ages. A sample of Dutch twin pairs was followed over a 13-year

period, and cognitive tests were conducted when the twins were 5, 7, 10, 12, and 18 years old. These data showed that genetic effects on verbal IQ become increasingly important with age, whilst shared environmental influences decrease. For nonverbal IQ, genetic effects show a modest increase with age, and shared environmental influences could not be detected. The stability of verbal and nonverbal abilities is mainly accounted for by genetic effects that exert their influence via a transmission structure. A shared environmental common factor structure is of moderate importance in explaining continuity in verbal abilities from age 5 to 10 years, but shared environmental influences are not important for stability in nonverbal abilities. Non-shared environmental influences exerted time specific influences only, and did not influence the stability of cognitive abilities. The overlap between verbal and nonverbal abilities is entirely accounted for by genetic effects, and this overlap increases slightly with age.

Genetic and environmental influences at different time points

The increase of genetic effects on verbal and nonverbal abilities with age is in accordance with findings from previous cross sectional and longitudinal studies into cognitive development (Deary et al., 2006; Petrill et al., 2004; Bartels et al., 2002; Bishop et al., 2003; Plomin & Spinath, 2004; Wilson, 1983). However, the current study is the first to cover cognitive development from childhood into young adulthood, and separates cognitive development into verbal and nonverbal abilities. We found the heritability of verbal IQ to increase from 46% at age 5 to 84% at age 18 years. For nonverbal IQ, genetic effects explained 64% of the variance at age 5. This proportion increased to 74% in young adulthood, but the confidence intervals of the heritability estimates at the

different ages overlap, and are therefore not significantly different from each other. The heritability estimates found at age 18 years are similar to the estimates reported in previous studies in young and middle-aged adults (Rijsdijk et al., 2002; Posthuma et al., 2001) that found a heritability of 84-85% for VIQ and of 68-69% for PIQ.

Shared environmental influences were found to only be of importance for individual differences in verbal abilities in childhood. The shared environment was not of importance for explaining variance in nonverbal IQ. In studies into the development of general cognitive abilities, some reported significant influences of shared environmental effects (Rietveld et al., 2003; Bartels et al., 2002; Bishop et al., 2003) whilst others failed to find significant effects of the shared environment (Petrill et al., 2004). The results of our study suggests that shared environmental influences are mainly important for verbal IQ, and less so for nonverbal aspects of cognitive performance. In accordance with previous studies (Bishop et al., 2003; Bartels et al., 2002), we found the shared environmental effects to decrease with age. Whilst shared environmental influences accounted for 28% of the variance at age 5 and age 7, these influences decreased to 6% at age 12 and became insignificant at age 18.

Longitudinal analyses: genetic and environmental effects on stability

Verbal and nonverbal abilities were found to be highly stable over time. Over a 13-year time interval the phenotypic correlations were around .50. The stability of nonverbal IQ was entirely accounted for by genetic effects. Stability of verbal IQ showed a moderate influence of shared environmental factors in early and middle childhood, but was entirely explained by genes in later phases of development. The major genetic effects

on stability of cognitive performance are in agreement with findings from prior longitudinal studies (Rietveld et al., 2003; Bartels et al., 2002; Petrill et al., 2004). Previous studies were inconclusive about the developmental mechanism underlying stability of cognitive development. Some studies found that a common factor structure gave a better description of the stability (Bartels et al., 2002; Petrill et al., 2004), whilst others reported a transmission structure (Cardon et al., 1992; Rietveld et al., 2003; Cardon, 1994) or a combination of both models (Bishop et al., 2003). Apart from Rietveld et al. (2003) and Cardon (1994), these previous studies examined general cognitive ability, and did not make a distinction into more specialized cognitive abilities. Similar to the latter two studies, in our project (which is a follow- up of the study of Rietveld et al., 2003) the underlying structure of genetic effects on stability was best described by a transmission model. This structure implies that, apart from substantial genetic effects that are carried over to continue to exert their influence on later time points, new genetic effects coming into play at subsequent time points are also of importance.

Shared environmental effects were found to have moderate effects on the stability of verbal abilities in early to middle childhood. These influences exerted their effects via a common factor structure loading at age 5, 7, and 10 years. In contrast, no shared environmental influences were found for nonverbal abilities. These findings indicate that children's development of verbal abilities is more prone to differences in the family environment than the development of nonverbal abilities. Factors such as socioeconomic status (SES) and parental education are highly stable and may underlie individual differences in verbal abilities in young children. Previous studies have shown that living

in a high-SES neighborhood is positively associated with IQ, verbal ability and reading ability in childhood and early adolescence, even when family characteristics associated with neighborhood characteristics are taken into account (Leventhal & Brooks-Gunn, 2000). Various researchers have attempted to specify the characteristics of the home environment that may be related to cognitive abilities. The HOME (Caldwell & Bradley, 2003) is one of the most widely used measures of the family environment. A recent review of studies using the HOME throughout the world (Bradley & Corwyn, 2005) suggested that the positive influence of learning stimulation provided by the parents on the development of cognitive abilities are strongest in early childhood. This is in line with our finding of decreasing shared environmental effects in later phases of childhood. Unfortunately, most studies exploring the association between environmental influences and cognition do not control for genetic influences on this association. One exception to this is a study by Petrill, Pike, Price & Plomin (2004), who did a twin study in early childhood and examined whether SES and chaos in the home mediate the shared environmental variance associated with cognitive abilities. They found that both measures mediated a significant but modest proportion of the shared environment. However, these effects were found to be similar for verbal and nonverbal abilities, whilst we only found significant shared environmental influences for verbal abilities. Several behavior genetics research groups have now started to include more precise measures of the shared environment in their data collection. Only be collecting such indices, will it be possible to gain more insight into shared environmental influences.

In agreement with previous studies, non-shared environmental influences were found to only be of importance for effects specific to each time point and did not

contribute to the continuity of cognitive abilities. Non-shared environmental influences are important in explaining why twins, and other children from the same family, are different from each other. Factors that may induce differences between twins and siblings could include traumatic experiences unshared with the co-twin, or consequences of an accident or illness. Also, if the children are in separate classes (as is the case for 37% of the twins, according to a large survey in 12-year-old twin pairs registered at the NTR), influences of the teacher will be non-shared. Within the Dutch primary school system, children normally change teacher each school year. The possible effects of a school teacher are thus likely to be age specific. Future studies should also include specific measures of non-shared environmental influences, in order to be able to examine the precise role of these effects on individual differences in cognitive abilities.

Genetic correlation between verbal and nonverbal abilities

Previous studies into the development of special cognitive abilities suggested that verbal and nonverbal abilities are largely genetically independent in early childhood, but become increasingly dependent in later phases of development (Petrill, 1997; Petrill et al., 2001; Price et al., 2000; Rijsdijk et al., 2002). The phenotypic association between verbal and nonverbal IQ in our study showed a slight increase over time, from .33 at age 5 to .57 at age 18. The overlap between verbal and nonverbal IQ was entirely accounted for by genetic effects. Following the increase in phenotypic correlation, the genetic correlation between verbal and nonverbal abilities also increased over time, from .62 at age 5 to .76 at age 12 and .73 at age 18. The genetic correlations found in the current study are larger than the correlations reported by Rietveld et al (2003), who used the

same study sample when the twins were 5, 7, and 10 years old. In Rietveld's study, genetic modeling was done on the six subtest scores of the RAKIT. In the current study we used composite verbal and nonverbal IQ scores instead of subtest scores. Additionally, Rietveld et al. examined the significance of shared environmental influences on all subtests together and did not test whether the influences may only be significant for verbal subtests. Therefore, in their model, part of the overlap between verbal and nonverbal abilities was accounted for by shared environmental effects. As the influence of shared environmental effects was found to be non-significant for nonverbal abilities in the current study, the covariance between verbal and nonverbal IQ was entirely explained by genetic effects (i.e. the bivariate heritability between verbal and nonverbal ability was 100%). Therefore, the genetic correlation (which is the standardized bivariate heritability) is higher in the current study than in the study by Rietveld et al.

The genetic correlation found in our study at age 18 was similar to the correlation reported by Posthuma et al. (2001) in young and middle-aged adults. They found a genetic correlation of .65 between VIQ and PIQ. Studying several specific cognitive abilities in the CAP project, Alarcón et al. found an average genetic correlation of .48 in 12-year olds (Alarcón et al., 1998) and of .52 in 16-year olds (Alarcón et al., 1999). In 2-year-old twins, Price et al (2000) reported a genetic correlation of .30. These results, together with the findings from our study, suggest that the genetic correlation between specific cognitive abilities increases with age. A strong genetic correlation is often conceived as evidence for a biological basis of g and for the existence of generalist genes (Plomin & Spinath, 2002; Kovas & Plomin, 2006; Petrill, 1997). Our findings support

this hypothesis. However, the genetic correlations are significantly different from one at all ages, indicating that there is also substantial genetic variance in verbal abilities that is unshared with nonverbal abilities, and vice versa.

Further considerations

To our knowledge, this is the first twin study reporting on the development of verbal and nonverbal abilities spanning childhood to young adulthood. Studying cognitive development over a broad time span necessitates different measurements per age group, simply because the development of cognition over such a long time cannot be captured by one test. One of the difficulties that come along with this is that no distinction can be made between true changes in development and changes due to different measurement instruments. In our study, we used the same test at the first three measurement occasions, namely the RAKIT. On the fourth and the fifth measurement occasion the WISC-R and the WAIS-III was used. The concurrent validity of the RAKIT and the WISC-R is .86 (Pijl et al., 1984). The correlation between VIQ and PIQ scores as measured with the WISC compared to the WAIS are respectively .89 and .76 (Wechsler, 1981). Based on this, we feel that it is likely that the patterns reported here reflect true development.

One of the challenges of longitudinal studies is drop out bias. Because of its longitudinal nature, this study had to deal with drop-outs over the years. Up to the fourth measurement occasion, more than 90% of the original sample still participated. As the twins had reached adulthood by the fifth measurement occasion, by then the choice of participation was no longer made by the parents. Also, at this age many twins had full

time jobs or were enrolled in a study program. For many families, lack of time or difficulties to take leave was the prime reason to no longer take part. At age 18 the participation rate decreased to 58% and new families were recruited in order to obtain a sufficient sample size. Comparison of the subjects who continued participation and the families who dropped out revealed higher cognitive ability scores at age 5 in the subjects who continued participation. To correct for any bias this may cause on parameter estimates, the data analyses were performed on the raw data, so that longitudinally incomplete data could also be included (Little & Rubin, 2002).

We did not find sex differences in MZ and DZ twin resemblance for verbal and nonverbal IQ. This is in accordance with nearly all genetic studies of cognition. For example, two twin family studies that included larger sample sizes in adults (Posthuma et al., 2001) and young children (Spinath et al., 2003) did not find substantial sex differences in MZ and DZ twin correlations for verbal and nonverbal IQ either.

Twin correlations and heritability estimates may be affected by possible effects of gene environment correlation or gene environment interaction (Posthuma & De Geus, 2006; Plomin, DeFries, McClearn, & McGuffin, 2001; Van Leeuwen, Van den Berg, & Boomsma, in revision). Parents with above average cognitive abilities are likely to have children who show above average cognitive performance, based on genetic transmission. But these parents may also be more likely to provide cognitively stimulating materials and interactions with their children, and thus provide an advantageous environment (cultural transmission). If both phenomena are of importance these two forms of transmission induce a correlation between genes and environment. However, the recent study of Van Leeuwen et al. did not obtain evidence for cultural transmission in an

independent sample of 9-year-old twins and their sibs. On the other hand, a certain genetic make-up could show differential expressions in different environments (gene environment interaction). For instance, a child with a genetic predisposition for above average cognitive performance may benefit more from an advantageous environment than a child with genetic vulnerability for low cognitive abilities. One approach to test for gene environment interaction is to examine the association between sum and difference scores in MZ twins (Jinks & Fulker, 1970). Sum scores of MZ twin pairs reflect familial effects (either genetic or shared environmental), while absolute differences within these pairs can only be caused by non-shared environmental effects. An association between these scores would suggest that (provided that shared environmental effects are absent), people with a certain genotype may be more vulnerable to non-shared environmental effects than people carrying other genetic variants (see also Van Leeuwen et al., in revision, for an application of this method). We explored this association for both verbal and nonverbal IQ at all 5 ages, resulting in 10 correlations. None of these were found to be significant, apart from the correlation for VIQ at age 7 ($r = .256$, $p = .024$) which is not significant after Bonferroni correction for multiple testing (Stevens, 1996). These results yield no indication for strong gene environment interaction on verbal and nonverbal IQ.

Our longitudinal data provide support for strong genetic effects on the continuity of verbal and nonverbal abilities from early childhood to young adulthood. Shared environmental effects are only of importance for verbal abilities, and these effects are limited to young and middle childhood. Non-shared environmental effects are of moderate importance in explaining individual differences in both verbal and nonverbal IQ, these effects are only age specific. These results add to the growing body of literature

on the development of cognitive abilities that report that genetic effects are the driving force behind stability of cognitive abilities. Previous studies also agreed that environmental effects are mainly important in explaining why twins differ from each other, and are predominantly time specific. This latter finding is important, because it implies that, although environmental influences are of significant importance in explaining individual differences in cognitive abilities, these effects are mainly transient in nature. In our study, shared environmental effects were found to contribute significantly to the stability in cognitive abilities in early and middle childhood. This finding suggests that, even in a relatively egalitarian society like the Netherlands, variance in the family environment is of significant importance. However, the precise nature of the shared en non-shared environmental effects is still largely unknown. Fortunately, several research groups have now begun to a) measure specific genes by candidate gene studies, and b) include specific measures of the environment. These studies will greatly increase our knowledge about the precise role of genes and environment on the development of cognitive abilities.

References

Alarcón, M., Plomin, R., Corley, R., & DeFries, J. C. (2003). Multivariate parent-offspring analyses of specific cognitive abilities. In S.A. Petrill, R. Plomin, J. C. DeFries, & J. K. Hewitt (Eds.), *Nature, nurture, and the transition to early adolescence* (pp. 28-48). New York: Oxford University Press, Inc.

Alarcón, M., Plomin, R., Fulker, D. W., Corley, R., & DeFries, J. C. (1998). Multivariate path analysis of specific cognitive abilities data at 12 years of age in the Colorado Adoption Project. *Behavior Genetics, 28,* 255-264.

Alarcón, M., Plomin, R., Fulker, D. W., Corley, R., & DeFries, J. C. (1999). Molarity not modularity: Multivariate genetic analysis of specific cognitive abilities in parents and their 16-year-old children in the Colorado Adoption Project. *Cognitive Development, 14,* 175-193.

Bartels, M., Rietveld, M. J. H., Van Baal, G. C. M., & Boomsma, D. I. (2002). Genetic and environmental influences on the development of intelligence. *Behavior Genetics, 32,* 237-249.

Bishop, E. G., Cherny, S. S., Corley, R., Plomin, R., DeFries, J. C., & Hewitt, J. K. (2003). Development genetic analysis of general cognitive ability from 1 to 12 years in a sample of adoptees, biological siblings, and twins. *Intelligence, 31,* 31-49.

Bleichrodt, N., Drenth, P. J. D., Zaal, J. N., & Resing, W. C. M. (1984). *Revisie Amsterdamse Kinder Intelligentie Test [Revised Amsterdam Child Intelligence Test]*. Lisse, the Netherlands: Swets & Zeitlinger B.V.

Boomsma, D. I. (1993). Current status and future prospects in twin studies of the development of cognitive abilities, infancy to old age. In T.J.Bouchard, Jr. & P. Propping (Eds.), *Twins as a Tool of Behavioral Genetics* (pp. 67-82). Chichester: John Wiley & Sons.

Boomsma, D. I. & Molenaar, P. C. (1987). The genetic analysis of repeated measures. I. Simplex models. *Behavior Genetics, 17,* 111-123.

Boomsma, D. I., Orlebeke, J. F., & Van Baal, G. C. M. (1992). The Dutch Twin Register: growth data on weight and height. *Behavior Genetics, 22,* 247-251.

Boomsma, D. I. & Van Baal, G. C. M. (1998). Genetic influences on childhood IQ in 5- and 7-year-old Dutch twins. *Developmental Neuropsychology, Special Issue, 14,* 115-126.

Boomsma, D. I., Vink, J. M., Van Beijsterveldt, C. E. M., De Geus, E. J. C., Beem, A. L., Mulder, E. J. et al. (2002). Netherlands Twin Register: a focus on longitudinal research. *Twin Research, 5,* 401-406.

Bornstein, M. H. & Sigman, M. D. (1986). Continuity in mental development from infancy. *Child Development, 57,* 251-274.

Bouchard, T. J., Jr. & McGue, M. (2003). Genetic and environmental influences on human psychological differences. *Journal of Neurobiology, 54,* 4-45.

Bradley, R. H. & Corwyn, R. F. (2005). Caring for children around the world: A view from HOME. *International Journal of Behavioral Development, 29,* 468-478.

Caldwell, B. M. & Bradley, R. H. (2003). *Home Observation for Measurement of the Environment: Administration manual*. Little Rock, AR: Authors.

Cardon, L. R. (1994). Specific cognitive abilities. In J.C. DeFries, R. Plomin, & D. W. Fulker (Eds.), *Nature and nurture during middle childhood* (pp. 57-76). Cambridge, Massachusetts 02142, USA: Blackwell Publishers.

Cardon, L. R., Fulker, D. W., DeFries, J. C., & Plomin, R. (1992). Continuity and change in general cognitive ability from 1 to 7 years of age. *Developmental Psychology, 28,* 64-73.

Carroll, J. B. (1993). *Human cognitive abilities: A survey of factor-analytis studies*. New York: Cambridge University Press.

Casto, S. D., DeFries, J. C., & Fulker, D. W. (1995). Multivariate genetic analysis of Wechsler Intelligence Scale for Children--Revised (WISC-R) factors. *Behavior Genetics, 25,* 25-32.

Deary, I. J. (2001). Human intelligence differences: a recent history. *Trends in Cognitive Sciences, 5,* 127-130.

Deary, I. J., Spinath, F. M., & Bates, T. C. (2006). Genetics of intelligence. *European Journal of Human Genetics, 14,* 690-700.

Deary, I. J., Whiteman, M. C., Starr, J. M., Whalley, L. J., & Fox, H. C. (2004). The impact of childhood intelligence on later life: following up the Scottish mental surveys of 1932 and 1947. *Journal of Personality and Social Psychology, 86,* 130-147.

DeFries, J. C., Johnson, R. C., Kuse, A. R., McClearn, G. E., Polovina, J., Vandenberg, S. G. et al. (1979). Familial resemblance for specific cognitive abilities. *Behavior Genetics, 1,* 23-48.

DiLalla, L. F., Thompson, L. A., Plomin, R., Phillips, K., Fagan, J. F., Haith, M. M. et al. (1990). Infant Predictors of Preschool and Adult IQ: A Study of Infant Twins and Their Parents. *Developmental Psychology, 26,* 759-769.

Eaves, L. J., Long, J., & Heath, A. C. (1986). A theory of developmental change in quantitative phenotypes applied to cognitive development. *Behavior Genetics, 16,* 143-162.

Fulker, D. W., DeFries, J. C., & Plomin, R. (1988). Genetic influence on general mental ability increases between infancy and middle childhood. *Nature, 336,* 767-769.

Jinks, J. L. & Fulker, D. W. (1970). Comparison of the biometrical genetical, MAVA, and classical approaches to the analysis of human behavior. *Psychological Bulletin, 73,* 311-349.

Jirtle, R. L. & Skinner, M. K. (2007). Environmental epigenomics and disease susceptibility. *Nature Reviews Genetics, 8,* 253-262.

Jöreskog, K. G. (1970). Estimation and testing of simplex models. *British Journal of Mathematical and Statistical Psychology, 23,* 121-145.

Kovas, Y. & Plomin, R. (2006). Generalist genes: implications for the cognitive sciences. *Trends in Cognitive Sciences, 10,* 198-203.

Leventhal, T. & Brooks-Gunn, J. (2000). The neighborhoods they live in: the effects of neighborhood residence on child and adolescent outcomes. *Psychological Bulletin, 126,* 309-337.

Little, R. J. A. & Rubin, D. B. (2002). *Statistical analysis with missing data*. (2 ed.) New york: Wiley and Sons.

Martin, N. G. & Eaves, L. J. (1977). The genetical analysis of covariance structure. *Heredity, 38,* 79-95.

McGue, M. & Bouchard, T. J., Jr. (1989). Genetic and environmental determinants of information processing and special mental abilities: A twin analysis. In R.J.Sternberg (Ed.), *Advances in the psychology of human intelligence, Vol. 5* (pp. 7-45). Hillsdale, NJ, England: Lawrence Erlbaum Associates, Inc.

Neale, M. C., Boker, S. M., Xie, G., & Maes, H. H. (2006). *Mx: Statistical modeling*. (7th ed.) Richmond, VA 23298: VCU, Department of Psychiatry.

Pedersen, N. L., Plomin, R., Nesselroade, J. R., & McClearn, G. E. (1992). A quantitative genetic analysis of cognitive abilities during the second half of the life span. *Psychol.Sci., 3,* 346-353.

Petrill, S. A. (1997). Molarity versus modularity of cognitive functioning? A behavioral genetic perspective. *Current Directions in Psychological Science, 6,* 96-99.

Petrill, S. A., Lipton, P. A., Hewitt, J. K., Plomin, R., Cherny, S. S., Corley, R. et al. (2004). Genetic and environmental contributions to general cognitive ability through the first 16 years of life. *Developmental Psychology, 40,* 805-812.

Petrill, S. A., Pike, A., Price, T. S., & Plomin, R. (2004). Chaos in the home and socioeconomic status are associated with cognitive development in early childhood: Environmental mediators identified in a genetic design. *Intelligence, 32,* 445-460.

Petrill, S. A., Saudino, K. S., Wilkerson, B., & Plomin, R. (2001). Genetic and environmental molarity and modularity of cognitive functioning in 2-year-old twins. *Intelligence, 29,* 31-43.

Pijl, Y. J., Hofman, R. H., Bleichrodt, N., Resing, W. C. M., Lutje-Spelberg, H. C., De Bruijn, E. E. et al. (1984). *Vergelijkbaarheid van de WISC-R en de RAKIT*. Amsterdam: Research Instituut voor Onderwijs in het Noorden, Vrije Universiteit.

Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P. (2001). *Behavioural Genetics*. (4th ed.) New York: Worth Publishers.

Plomin, R. & Spinath, F. M. (2002). Genetics and general cognitive ability (g). *Trends in Cognitive Sciences, 6,* 169-176.

Plomin, R. & Spinath, F. M. (2004). Intelligence: genetics, genes, and genomics. *Journal of Personality and Social Psychology, 86,* 112-129.

Posthuma, D., Baare, W. F. C., Hulshoff Pol, H. E., Kahn, R. S., Boomsma, D. I., & De Geus, E. J. C. (2003). Genetic correlations between brain volumes and the WAIS-

III dimensions of verbal comprehension, working memory, perceptual organization, and processing speed. *Twin Research, 6,* 131-139.

Posthuma, D. & De Geus, E. J. C. (2006). Progress in the Molecular-Genetic Study of Intelligence. *Current Directions in Psychological Science, 15,* 151-155.

Posthuma, D., De Geus, E. J. C., & Boomsma, D. I. (2001). Perceptual speed and IQ are associated through common genetic factors. *Behavior Genetics, 31,* 593-602.

Price, T. S., Eley, T. C., Dale, P. S., Stevenson, J., Saudino, K., & Plomin, R. (2000). Genetic and environmental covariation between verbal and nonverbal cognitive development in infancy. *Child Development, 71,* 948-959.

Rice, T., Carey, G., Fulker, D. W., & DeFries, J. C. (1989). Multivariate path analysis of specific cognitive abilities in the Colorado Adoption Project: Conditional path model of assortative mating. *Behavior Genetics, 19,* 195-207.

Rietveld, M. J. H., Dolan, C. V., Van Baal, G. C. M., & Boomsma, D. I. (2003). A twin study of differentiation of cognitive abilities in childhood. *Behavior Genetics, 33,* 367-381.

Rietveld, M. J. H., Van Baal, G. C. M., Dolan, C. V., & Boomsma, D. I. (2000). Genetic factor analyses of specific cognitive abilities in 5-year-old Dutch children. *Behavior Genetics, 30,* 29-40.

Rijsdijk, F. V., Vernon, P. A., & Boomsma, D. I. (2002). Application of hierarchical genetic models to Raven and WAIS subtests: a Dutch twin study. *Behavior Genetics, 32,* 199-210.

Scarr, S. & Weinberg, R. A. (1983). The Minnesota Adoption Studies: genetic differences and malleability. *Child Development, 54,* 260-267.

Spinath, F. M., Ronald, A., Harlaar, N., Price, T. S., & Plomin, R. (2003). Phenotypic g early in life: On the etiology of general cognitive ability in a large population sample of twin children aged 2-4 years. *Intelligence, 31,* 195-210.

Stevens, J. (1996). *Multivariate statistics for the social sciences*. (3rd ed.) Mahwah, New Jersey: Lawrence Erlbaum Associates, Inc.

Van Baal, G. C. M., Boomsma, D. I., & De Geus, E. J. C. (2001). Longitudinal genetic analysis of EEG coherence in young twins. *Behavior Genetics, 31,* 637-651.

Van Haassen, P. P., De Bruijn, E. E., Pijl, Y. J., Poortinga, Y. H., Lutje-Spelberg, H. C., Vander Steene, G. et al. (2006). *Wechsler Intelligence Scale for Children-Revised, Dutch Version*. Lisse, the Netherlands: Swets & Zetlinger B.V.

Van Leeuwen, M., Van den Berg, S. M., & Boomsma, D. I. A twin-family study of general IQ. *Learning and Individual Differences,* (in revision).

Wechsler, D. (1981). *The Wechsler Adult Intelligence Scale-Revised*. New York: Psychological Corporation.

Wechsler, D. (1997). *Wechsler Adult Intelligence Scale-Third edition, Dutch Version*. Lisse, the Netherlands: Swets & Zeitlinger B.V.

Wilson, R. S. (1983). The Louisville Twin Study: developmental synchronies in behavior. *Child Development, 54,* 298-316.

Wilson, R. S. (1986). Continuity and change in cognitive ability profile. *Behavior Genetics, 16,* 45-60.

Table 1. Descriptives of the verbal and nonverbal IQ scores for all subjects at all time points (age 5, 7, 10, 12, and 18 years)

All twins	N	Mean	SD
Verbal IQ age 5	415	103.79	13.47
Nonverbal IQ age 5	418	101.17	13.75
Verbal IQ age 7	382	97.68	14.22
Nonverbal IQ age 7	384	107.25	16.10
Verbal IQ age 10	392	103.09	14.90
Nonverbal IQ age 10	394	108.68	16.33
Verbal IQ age 12	381	97.33	12.96
Nonverbal IQ age 12	383	103.10	14.05
Verbal IQ age 18	365	101.02	19.36
Nonverbal IQ age 18	364	107.12	17.01

Table 2. Longitudinal phenotypic correlations for verbal abilities (above diagonal) and nonverbal abilities (below diagonal) and the cross-sectional correlations between verbal and nonverbal abilities at age 5, 7, 10, 12 and 18 years

age	**5**	**7**	**10**	**12**	**18**	**$R_{v\text{-}nv}$**
5	-	.64	.61	.55	.51	.33
7	.58	-	.61	.56	.55	.35
10	.57	.71	-	.68	.67	.35
12	.54	.62	.66	-	.80	.58
18	.47	.57	.63	.61	-	.57

Table 3. Twin correlations for verbal and nonverbal abilities in all zygosity groups

	Verbal abilities					Nonverbal abilities				
age	**5**	**7**	**10**	**12**	**18**	**5**	**7**	**10**	**12**	**18**
MZM	.75	.56	.84	.86	.88	.71	.60	.73	.85	.76
DZM	.62	.57	.51	.65	.42	.46	.38	.58	.48	.31
MZF	.81	.70	.80	.87	.81	.56	.71	.70	.77	.74
DZF	.65	.73	.33	.59	.52	.43	.30	.40	.57	.44
DOS	.63	.55	.50	.56	.38	.65	.53	.42	.49	.57
All MZ	.77	.61	.82	.86	.83	.61	.68	.71	.81	.73
All DZ	.59	.58	.42	.59	.41	.49	.42	.45	.45	.39

Note: MZM = monozygotic male twin pairs; DZM = dizygotic male twin pairs; MZF = monozygotic female twin pairs; DZF = dizygotic female twin pairs; DOS = dizygotic opposite sex twin pairs; All MZ = all monozygotic twin pairs; All DZ = all dizygotic twin pairs.

Table 4. Cross-twin/cross-age correlations over time for verbal and nonverbal abilities in monozygotic (MZ, above diagonal) and dizygotic (DZ, below diagonal) twins, and the cross-twin/cross-trait correlations between verbal and nonverbal abilities in MZ (first number) and DZ (second number) twins at age 5, 7, 10, 12 and 18 years.

	Verbal abilities						**Nonverbal abilities**					**Cross $R_{v\text{-}nv}$**
age	**5**	**7**	**10**	**12**	**18**	**age**	**5**	**7**	**10**	**12**	**18**	**MZ/DZ**
5	-	.59	.58	.55	.42	**5**	-	.54	.57	.56	.58	.32/.32
7	.48	-	.61	.55	.50	**7**	.37	-	.66	.60	.62	.28/.21
10	.40	.40	-	.65	.65	**10**	.33	.35	-	.63	.65	.32/.27
12	.41	.37	.41	-	.79	**12**	.37	.36	.38	-	.65	.53/.37
18	.36	.33	.35	.43	-	**18**	.23	.25	.34	.37	-	.53/.28

Table 5. Model fitting results for multivariate longitudinal analyses of verbal and nonverbal abilities

model		-2LL	df	Cpm	χ^2	p	AIC
1.	ACE Cholesky	29270.163	3693				
2.	ACE A common + age specific C common + age specific E age specific only	29403.336	3806	1	133.173	.095	-92.827
3.	ACE A transmission C common + age specific E age specific only	29392.248	3804	1	122.085	.222	-99.915
4.	ACE A transmission C common verbal only C age specific verbal + nonverbal E age specific only	29402.021	3810	3	9.773	.135	
5.	ACE A transmission C common verbal only C age specific verbal only E age specific only	29405.304	3815	4	3.283	.656	
6.	ACE A transmission C common verbal only, no age 18 C age specific verbal only E age specific only	29406.790	3816	5	1.486	.223	
7.	ACE A transmission C common verbal only, no age 18, 12 C age specific verbal only E age specific only	29408.735	3817	6	1.945	.163	
8.	ACE A transmission C common verbal only, no age 18, 12, 10 C age specific verbal only E age specific only	29427.400	3818	7	18.665	<.001	
9.	**ACE** **A transmission** **C common verbal only, no age 18, 12** **C age specific verbal only, no age 18** **E age specific only**	**29408.735**	**3818**	**7**	**0**	**1.00**	
10.	ACE A transmission C common verbal only, no age 18, 12 C age specific verbal only, no age 18, 12 E age specific only	29413.496	3819	9	4.761	.029	

Note: -2LL = -2 log likelihood; df = degrees of freedom; cpm = compared to model

Table 6. Parameter estimates for additive genetic, shared environmental, and non-shared environmental influences as derived from the Cholesky decomposition.

		Verbal abilities					Nonverbal abilities				
	age	**5**	**7**	**10**	**12**	**18**	**5**	**7**	**10**	**12**	**18**
		Cholesky parameter estimates additive genetic effects									
Verbal	**5**	8.05									
	7	6.21	4.37								
	10	8.17	7.62	3.62							
	12	6.20	5.77	-1.50	4.10						
	18	7.22	12.18	-1.77	8.01	2.02					
Nonverbal	**5**	1.74	4.55	-4.43	.02	2.50	2.77				
	7	3.76	5.05	-5.83	1.60	5.80	5.81	.01			
	10	3.39	5.29	-6.40	2.89	5.52	5.27	.01	.00		
	12	4.18	5.49	-4.36	.04	.71	7.94	.03	.00	.01	
	18	2.80	8.31	-7.77	.01	4.39	5.95	.01	.00	.00	.00
		Cholesky parameter estimates shared environmental effects									
Verbal	**5**	8.62									
	7	7.53	4.92								
	10	5.52	.34	2.72							
	12	5.19	-.20	3.43	4.05						
	18	6.56	-.67	1.68	1.53	1.13					
Nonverbal	**5**	5.46	-3.12	1.60	-1.99	-4.00	.04				
	7	3.14	.12	3.11	.76	-3.94	.05	.00			
	10	3.73	-2.24	4.95	-1.07	.13	.00	.00	.00		
	12	4.10	-2.77	2.34	1.07	-.73	.02	.00	.00	.00	
	18	4.13	-1.85	1.09	.74	2.52	-.02	.00	.00	.00	.00
		Cholesky parameter estimates non-shared environmental effects									
Verbal	**5**	6.40									
	7	1.28	8.09								
	10	.97	-.03	6.44							
	12	.02	.29	.56	4.80						
	18	2.59	.88	.59	1.55	6.99					
Nonverbal	**5**	-.03	-.13	-.44	-.15	-.29	8.21				
	7	-.39	1.34	1.04	-.16	2.87	.95	8.37			
	10	-.89	.87	.90	.37	.79	.73	1.43	8.41		
	12	-.45	.57	.34	1.44	.30	-.44	-.02	.40	6.30	
	18	-1.19	1.07	.81	.89	2.52	-.45	-.59	-.47	-2.37	7.94

Table 7. Contributions of additive genetic (A), shared (C) and non-shared (E) environmental influences to the variance in verbal and nonverbal abilities at age 5, 7, 10, 12 and 18 years, based on the best fitting model (95% confidence intervals in parentheses)

	Verbal abilities					Nonverbal abilities	
Variance	**A transmission**	**C total**	**C common factor**	**C age specific**	**E age specific**	**A transmission**	**E age specific**
5	.46 (.33-.59)	.28 (.16-.40)	.20	.08	.26 (.20-.34)	.64 (.54-.72)	.36 (.28-.46)
7	39 (.29-.49)	.28 (.18-.38)	.26	.02	.33 (.26-.42)	.68 (.61-.75)	.32 (.25-.39)
10	.56 (.48-.65)	.16 (.09-.24)	.08	.08	.28 (.21-.35)	.69 (.62-.76)	.31 .24-.38)
12	.80 (.73-.86)	.06 (.01-.12)	-	.06	.14 (.11-.19)	.74 (.65-81)	.26 (.19-.35)
18	.84 (.78-.88)	-	-	-	.16 (.12-.22)	.74 (.65-.80)	.26 (.20-.35)

Table 8. Contributions of additive genetic (A), shared (C), and nonshared (E) environmental influences to the covariance in verbal and nonverbal abilities over time based on the best fitting model (95% confidence intervals in parentheses*)

	Verbal abilities			Nonverbal abilities	
Covariance	**A transmission**	**C common factor**	**E age specific**	**A transmission**	**E age specific**
5-7	.63 (.50-.77)	.37 (.23-.50)	-	1	-
5-10	.78 (.66-.89)	.22 (.11-.34)	-	1	-
5-12	1	-	-	1	-
5-18	1	-	-	1	-
7-10	.76 (.65-.86)	.24 (.14-.35)	-	1	-
7-12	1	-	-	1	-
7-18	1	-	-	1	-
10-12	1	-	-	1	-
10-18	1	-	-	1	-
12-18	1	-	-	1	-

* In the cases in which there is only one component specified to account for the covariance over time, this influence is per definition 100%. Therefore, the confidence intervals cannot be estimated in these cases.

Table 9. Genetic correlation (95% confidence intervals in parentheses) between verbal and nonverbal abilities at age 5, 7, 10, 12 and 18 years

age	R_g
5	.62 (.54- .74)
7	.67 (.59- .79)
10	.57 (.52- .63)
12	.76 (.71- .81)
18	.73 (.69- .78)

Figure captions.

Figure 1a.

Common factor model with age specific influences. Note, V5/NV5 = Verbal /Nonverbal abilities at age 5; A_v/A_{nv} = Common genetic factor exerting its influence on verbal /nonverbal abilities; A_s = age specific genetic influences. Path diagram is shown for additive genetic effects. The same model was also tested for shared environmental effects.

Figure 1b.

Transmission model. Note, V5/NV5 = Verbal /Nonverbal abilities at age 5; A_v/A_{nv} = Genetic influences on verbal /nonverbal abilities; ξA = genetic innovation; β = genetic transmission to subsequent time point

Figure 2.

Path diagram of the best fitting model. Note, V5/NV5 = Verbal /Nonverbal abilities at age 5; A_v/A_{nv} = Genetic influences on verbal /nonverbal abilities; ξA = genetic innovation; β = genetic transmission to subsequent time point; C_v = shared environmental common factor influencing verbal abilities; C_s = age specific shared environmental influences; E_s = age specific non-shared environmental influences.

FIGURE 1a

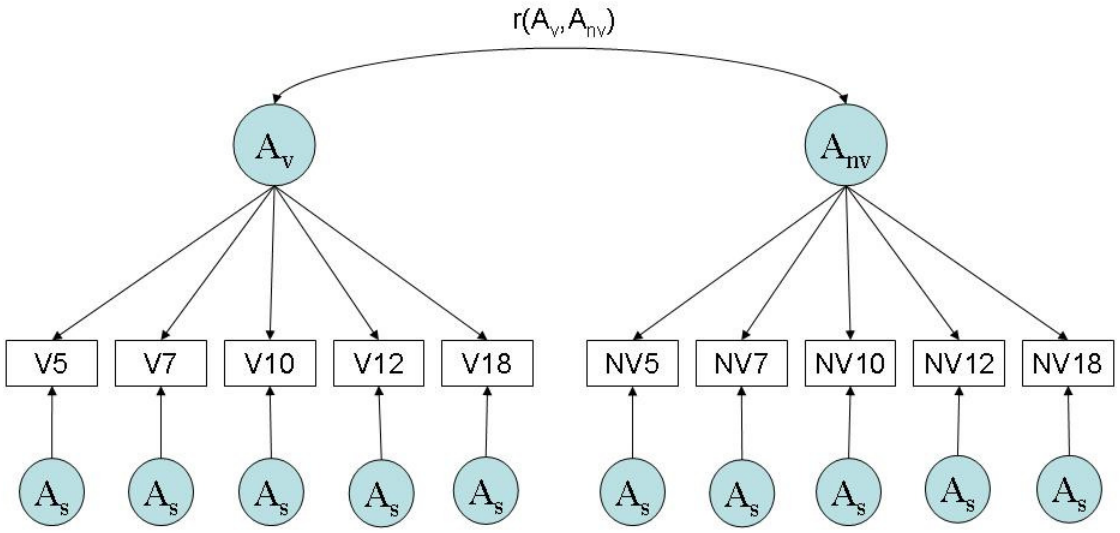


FIGURE 1b

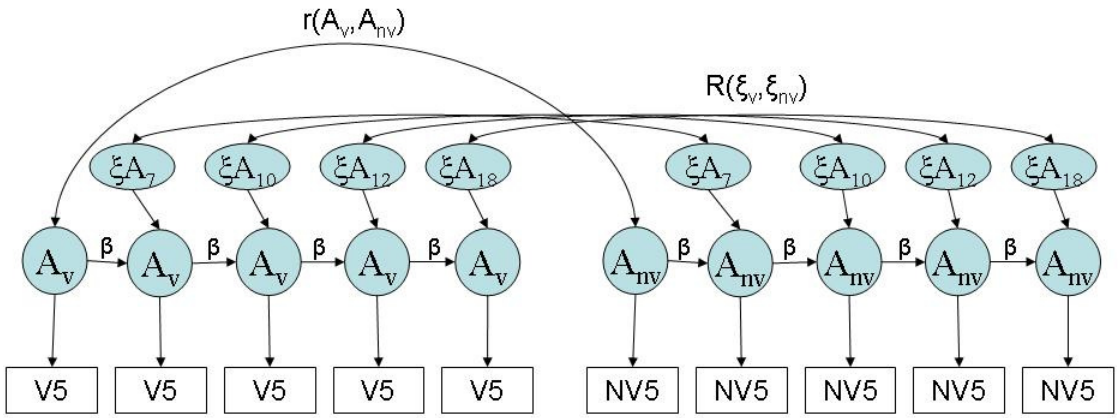
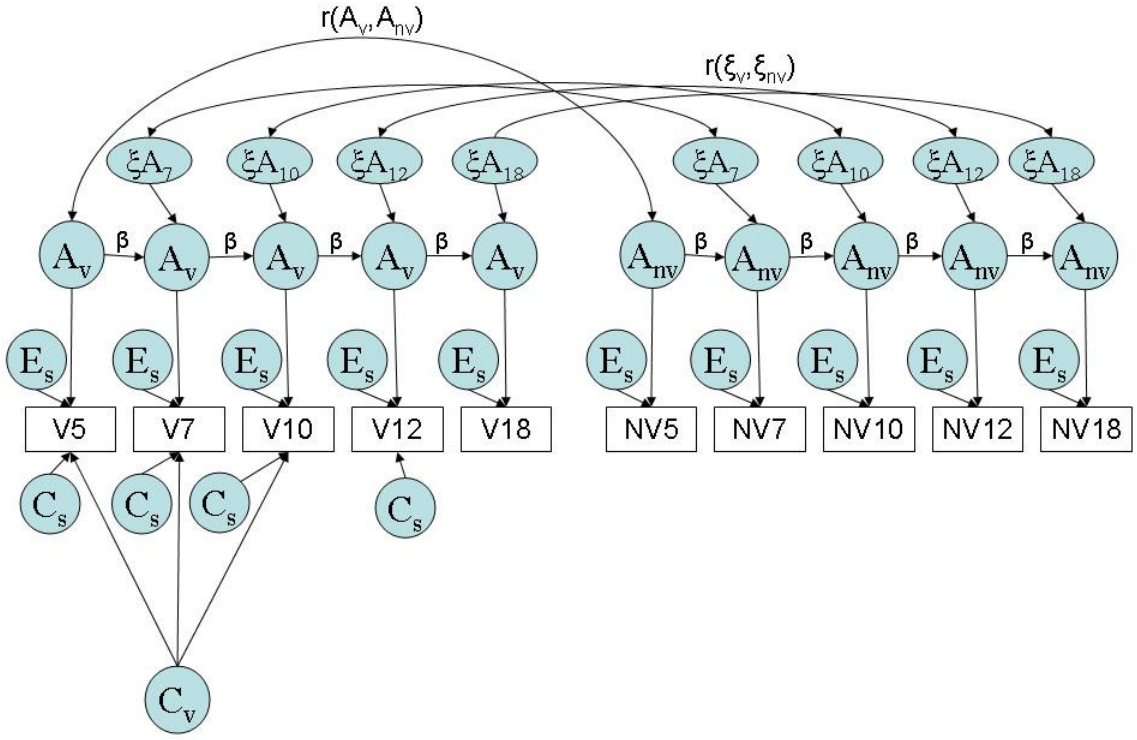


FIGURE 2


r(Av, Anv)
r(ξv, ξnv)
ξA7
ξA10
ξA12
ξA18
ξA7
ξA10
ξA12
ξA18
Av
β
Av
β
Av
β
Av
β
Av
Anv
β
Anv
β
Anv
β
Anv
β
Anv
Es
Es
Es
Es
Es
Es
Es
Es
Es
Es
V5
V7
V10
V12
V18
NV5
NV7
NV10
NV12
NV18
Cs
Cs
Cs
Cs
Cv